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FILE LAST UPDATED: 22 Apr 2010  (20100422/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Feb 2010
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Feb 2010

CAplus now includes complete International Patent Classification (IPC)
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=> s (?PARKINSON?(W)?PLUS?(W)?SYNDROM?) and (?PARKINSON? OR ALZHEIMER? OR
?PROGRES?(W)?SUPRANUCLEAR?(W)?PALSY? OR ?CORTICOBAS?(W)?DEGENERAT? OR
?DIFFUSE?(W)?DEMENT? OR ?LEWY?(W)?BODIES?)
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        228694 ?PLUS?
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=> d l1 ibib abs hitstr 1-18

L1 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2010:112032 CAPLUS
DOCUMENT NUMBER: 152:207144
TITLE: Tissue kallikrein for the treatment of Parkinson's disease, dementia with Lewy bodies, and related conditions
INVENTOR(S): Charles, Matthew L.; Williams, Mark
PATENT ASSIGNEE(S): Sanomune Inc., Can.
SOURCE: PCT Int. Appl., 48pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2010009557	A1	20100128	WO 2009-CA1051	20090724

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2008-83650P P 20080725
AB The invention discloses methods for treating Parkinson's disease, dementia with Lewy bodies, and conditions associated with Parkinson's disease and dementia with Lewy bodies. The methods include administering a therapeutically effective amount of tissue kallikrein, variants or active fragments thereof.
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2009:1193760 CAPLUS
DOCUMENT NUMBER: 151:378550
TITLE: Genetic variants of the α-synuclein gene SNCA are associated with Multiple system atrophy
AUTHOR(S): Al-Chalabi, Ammar; Durr, Alexandra; Wood, Nicholas W.; Parkinson, Michael H.; Camuzat, Agnes; Hulot, Jean-Sebastien; Morrison, Karen E.; Renton, Alan; Sussmuth, Sigurd D.; Landwehrmeyer, Bernhard G.; Ludolph, Albert; Agid, Yves; Brice, Alexis; Leigh, P. Nigel; Bensimon, Gilbert
CORPORATE SOURCE: NNIPPS Genetic Study Group, MRC Centre for Neurodegeneration Research, Department of Clinical Neuroscience, Institute of Psychiatry, and NIHR Biomedical Research Centre, King's College London, London, UK
SOURCE: PLoS One (2009), 4(9), No pp. given
CODEN: POLNCL; ISSN: 1932-6203
URL: http://www.plosone.org/article/fetchObjectAttachm

ent.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0007114&representation=PDF
PUBLISHER: Public Library of Science
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Background: Multiple system atrophy (MSA) is a progressive neurodegenerative disorder characterized by parkinsonism, cerebellar ataxia and autonomic dysfunction. Pathogenic mechanisms remain obscure but the neuropathol. hallmark is the presence of α-synuclein-immunoreactive glial cytoplasmic inclusions. Genetic variants of the α-synuclein gene, SNCA, are thus strong candidates for genetic association with MSA. One follow-up to a genome-wide association of Parkinson's disease has identified association of a SNP in SNCA with MSA. Methodol./Findings: We evaluated 32 SNPs in the SNCA gene in a European population of 239 cases and 617 controls recruited as part of the Neuroprotection and Natural History in Parkinson Plus Syndromes (NNIPPS) study. We used 161 independently collected samples for replication. Two SNCA SNPs showed association with MSA: rs3822086 (P = 0.0044), and rs3775444 (P = 0.012), although only the first survived correction for multiple testing. In the MSA-C subgroup the association strengthened despite more than halving the number of cases: rs3822086 P = 0.0024, OR 2.153, (95% CI 1.3-3.6); rs3775444 P = 0.0017, OR 4.386 (95% CI 1.6-11.7). A 7-SNP haplotype incorporating three SNPs either side of rs3822086 strengthened the association with MSA-C further (best haplotype, P = 8.7 + 10-4). The association with rs3822086 was replicated in the independent samples (P = 0.035). Conclusions/Significance: We report a genetic association between MSA and α-synuclein which has replicated in independent samples. The strongest association is with the cerebellar subtype of MSA.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2009:255925 CAPLUS
DOCUMENT NUMBER: 151:310606
TITLE: Reduced NADH coenzyme Q dehydrogenase activity in platelets of Parkinson's disease, but not Parkinson plus patients, from an Indian population
AUTHOR(S): Varghese, Merina; Pandey, Mritunjay; Samanta, Ananda; Gangopadhyay, Prasanta Kumar; Mohanakumar, Kochupurackal P.
CORPORATE SOURCE: Division of Cell Biology and Physiology, Laboratory of Clinical & Experimental Neuroscience, Indian Institute of Chemical Biology (CSIR), West Bengal, 700032, India
SOURCE: Journal of the Neurological Sciences (2009), 279(1-2), 39-42
CODEN: JNSCAG; ISSN: 0022-510X
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The observation of decline in mitochondrial electron transport chain function, specifically at complex I, in patients with Parkinson's disease (PD) has been reported by several groups. This study investigates whether a defect of mitochondrial function is present in the platelets of PD patients from an Indian population. We found that the NADH dehydrogenase activity in the platelets of PD patients is lower than that in healthy age- and gender-matched controls, while the succinate dehydrogenase activity was similar in both groups. Furthermore, there was no change in either of the activities in patients with Parkinson plus syndrome or atypical parkinsonism. This

is the first report indicating a decline in mitochondrial function in the platelets of PD patients from the Indian population, offering further support to the role of a mitochondrial defect in PD.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2009:53305 CAPLUS

DOCUMENT NUMBER: 150:98665

TITLE: Preparation of phenylalanine derivatives having dopaminergic activity

INVENTOR(S): Hobbs, Christopher

PATENT ASSIGNEE(S): Proximagen Ltd., UK

SOURCE: PCT Int. Appl., 50pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009007696	A1	20090115	WO 2008-GB2313	20080704
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2008273923	A1	20090115	AU 2008-273923	20080704
CA 2692608	A1	20090115	CA 2008-2692608	20080704
EP 2176217	A1	20100421	EP 2008-775861	20080704
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, AL, BA, MK, RS				

PRIORITY APPLN. INFO.: GB 2007-13189 A 20070706
WO 2008-GB2313 W 20080704

OTHER SOURCE(S): CASREACT 150:98665; MARPAT 150:98665

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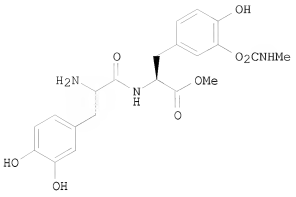

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AB The invention relates to phenylalanine derivs. (S)-RNHCH(CO2H)CH2C6H3(OR1)(OR2)-3,4 or their esters, amides, or salts [R is H or a natural or non-natural α-amino acid residue; one of R1 and R2 is (un)substituted aminocarbonyl or aminosulfonyl and the other is alkanoyl, fluoroalkanoyl, or cyclopropylcarbonyl], which diminish the symptoms of dopamine deficiency. Thus, peptide I.HCl was prepared by a multistep sequence starting from (S)-2-(tert-butoxycarbonylamino)-3-(3,4-dihydroxyphenyl)propionic acid Me ester. I.HCl showed increased locomotor activity, compared to baseline, in 6-OHDA-lesioned rats. All example compds. of the invention were shown to be converted in vivo to L-dopa to varying extents and over varying periods of time.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2008:261053 CAPLUS

DOCUMENT NUMBER: 148:441073

TITLE: Investigating the dopaminergic synapse in vivo. I. Molecular imaging studies in humans

AUTHOR(S): Nikolaus, Susanne; Antke, Christina; Kley, Konstantin; Poeppel, Thorsten D.; Hautzel, Hubertus; Schmidt, Daniela; Mueller, Hans-Wilhelm

CORPORATE SOURCE: Clinic of Nuclear Medicine, University Hospital Duesseldorf, Duesseldorf, Germany

SOURCE: Reviews in the Neurosciences (London, United Kingdom) (2007), 18(6), 439-472
CODEN: RNEUEO; ISSN: 0334-1763

PUBLISHER: Freund & Pettman

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Dopaminergic synaptic function may be assessed either at the presynaptic terminal or at the postsynaptic binding sites using mol. in vivo imaging methods. Apart from the d. of binding sites, parameters such as alterations in dopamine synthesis, dopamine storage or dopamine release can be quantified either by application of specific radiotracers or by assessing the competition between the exogenous radioligand and endogenous dopamine. Investigations of humans in both clin. and exptl. settings have yielded evidence that disturbances of dopaminergic function may be associated with numerous neurol. and psychiatric conditions, among which are movement disorders, schizophrenia, attention-deficit hyperactivity disorder, depression and drug abuse. This article gives an overview of those studies, which so far have been performed on dopaminergic neurotransmission in humans using in vivo imaging methods. We focus on disease-related deficiencies within the functional entity of the dopaminergic synapse. Taken together, in vivo findings yield evidence of

presynaptic dysfunctions in Parkinson's disease with decreases in striatal dopamine synthesis, dopamine storage, dopamine release and dopamine transporter binding. In contrast, 'Parkinson plus' syndromes (multiple system atrophy, progressive supranuclear palsy, cortico-basal degeneration, dementia with Lewy bodies) are characterized by both pre- and postsynaptic deficiencies with redns. in striatal dopamine synthesis, dopamine storage, dopamine release, and dopamine transporter, as well as D1 and D2 receptor binding. In patients with Huntington's disease, postsynaptic dysfunctions with redns. of striatal D1 and D2 receptor binding have become apparent, whereas attention-deficit/hyperactivity disorder is mainly characterized by presynaptic deficits with increases in dopamine transporter binding. Interestingly, findings are also consistent with respect to drug abuse: cocaine, amphetamine, methylphenidate, heroin, alc. and nicotine invariably act via enhancement of dopamine release in dorsal and/or ventral striatal regions. In vivo findings addnl. suggest that not only D2 receptor binding but also the extent of dopamine release is lower in individuals with a history of drug abuse. Findings become inconsistent with increasing complexities of psychiatric conditions. As yet, there is no clear evidence as to the contributions of the individual presynaptic and postsynaptic constituents of the dopaminergic synapse to the pathophysiologies of schizophrenia and depression. As these diseases can be conceived as the result of a variety of dysfunctions and dysregulations within an intricate network of neurotransmitter systems, regional investigations of one single pre- or postsynaptic constituent may not reach far enough to disentangle the interrelationships between the constituents of one let alone a variety of neurotransmitter systems.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

REFERENCE COUNT: 377 THERE ARE 377 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2007:1007158 CAPLUS

DOCUMENT NUMBER: 147:362330

TITLE: Pathological study on nigrostriatum in patients with Parkinson's disease and Parkinsonism plus syndrome

AUTHOR(S): Zhu, Mingwei; Wang, Luning; Luo, Yi; Wang, Zhenfu; Hu, Yazhuo

CORPORATE SOURCE: Department of Geriatric Neurology, General Hospital of Chinese PLA, Beijing, 100853, Peop. Rep. China

SOURCE: Zhonghua Shenjingke Zazhi (2006), 39(4), 250-254
CODEN: ZSZAFN; ISSN: 1006-7876

PUBLISHER: Zhonghua Yixuehui Zazhishe

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The histopathol. features in the nigrostriatal tissues of Parkinson's disease (PD) and Parkinsonism plus syndrome were explored. The substantia nigra and the striatum of 5 PD cases, 3 progressive supranuclear palsy (PSP) cases, 3 multiple system atrophy (MSA) cases and 5 normal aging control cases were examined by routine neuropathol. methods, Gallyas-Braak staining, tau, ubiquitin and α-synuclein immunohistochem. staining. Pigmented neurons in the substantia nigra of PD, PSP, MSA and the normal control cases were counted. The neuronal and glial cytoplasmic inclusions in the nigrostriatal tissues were observed The components of the abnormal proteins were identified. Nerve cells in the substantia nigra of PD, PSP and MSA groups showed severe loss in number, especially the ventrolateral zone and

the ventromedial zone. Compared with those in the normal aging control group, the nos. of nerve cells in the ventrolateral ones of PD, PSP and MSA groups decreased to 37.5%, 24.2% and 33.8% in the right side, and 48.0%, 25.8% and 33.9% in the left side, resp. There were α-synuclein and ubiquitin-pos. lewy bodies in the substantia nigra of PD. A lot of tau-pos., argyrophilic globous neurofibrinary tangles, tuft-shaped astrocytes and coiled bodies in the substantia nigra and the striatum of PSP were observed Severe loss of neurons and gliosis in the caudate nucleus and putamen of MSA were found. In addition, α-synuclein and ubiquitin-pos. glial cytoplasmic inclusions were found in the substantia nigra and striatal region of MSA. In conclusion, Lewy bodies in PD and glial cytoplasmic inclusions in MSA were related to abnormal depositions of α-synuclein and ubiquitin. Neuronal and glial cytoplasmic inclusions in PSP were related to abnormal aggregation of tau.

L1 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2006:1226630 CAPLUS
DOCUMENT NUMBER: 146:775
TITLE: Use of human and rat osteopontin in treatment of Parkinson's disease and other neurodegenerative disorders
INVENTOR(S): Jenner, Peter; Iczkiewicz, Joanna
PATENT ASSIGNEE(S): Proximagen Ltd., UK
SOURCE: U.S. Pat. Appl. Publ., 25pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20060264371	A1	20061123	US 2006-356453		20060217
PRIORITY APPLN. INFO.:			US 2005-653959P	P	20050218

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The present invention relates to the use of human and rat osteopontin (OPN) in treatment of Parkinson's disease and other neurodegenerative disorders especially those associated with aging. In particular, the inventors have demonstrated that intranigral injection of lipopolysaccharide (LPS) induces rapid and marked gliosis that accompanies loss of TH-pos. neurons and suggests that following glial cell activation, there is enhanced expression of OPN linked to increased. The inventors have also demonstrated that administration of anti-osteopontin antibody can induce dopaminergic or tyrosine hydroxylase neuron degeneration in a dose-dependent manner, indicating that endogenous OPN has a role to play in preventing neurodegeneration. The inventors have also shown that exogenous OPN has no effect on the. They have also demonstrated that the neuroprotective effects of OPN are not mediated via av53 integrin receptors.

L1 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2005:1075628 CAPLUS
DOCUMENT NUMBER: 143:319182
TITLE: Use of rotigotine for treating and preventing Parkinson-plus syndrome
INVENTOR(S): Scheller, Dieter
PATENT ASSIGNEE(S): Schwarz Pharma A.-G., Germany
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005092331	A1	20051006	WO 2005-EP3013	20050322
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 102004014841	A1	20051013	DE 2004-102004014841	20040324
DE 102004014841	B4	20060706		
AU 2005226911	A1	20051006	AU 2005-226911	20050322
CA 2559683	A1	20051006	CA 2005-2559683	20050322
EP 1727539	A1	20061206	EP 2005-728686	20050322
EP 1727539	B1	20071031		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				
CN 1960724	A	20070509	CN 2005-80009165	20050322
JP 2007530484	T	20071101	JP 2007-504337	20050322
AT 376828	T	20071115	AT 2005-728686	20050322
ES 2296156	T3	20080416	ES 2005-728686	20050322
MX 2006010747	A	20061215	MX 2006-10747	20060920
US 20070191470	A1	20070816	US 2006-593964	20060922
KR 2006130730	A	20061219	KR 2006-721135	20061011
NO 2006004792	A	20061023	NO 2006-4792	20061023
HK 1100765	A1	20080125	HK 2007-105904	20070605

PRIORITY APPLN. INFO.: DE 2004-102004014841A 20040324
WO 2005-EP3013 W 20050322

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention discloses the use of rotigotine, or a salt or prodrug thereof, as a medicament for preventing and/or treating Parkinson -plus syndrome.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 9 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2005:274998 CAPLUS
DOCUMENT NUMBER: 143:283383
TITLE: Heredofamilial parkinsonism
AUTHOR(S): Pal, Pramod Kumar; Wszolek, Zbigniew K.
CORPORATE SOURCE: National Institute of Mental Health and Neurosciences, Bangalore, India
SOURCE: Parkinson's Disease (2005), 139-158. Editor(s): Ebadi, Manuchair; Pfeiffer, Ronald E. CRC Press LLC: Boca Raton, Fla.
CODEN: 69GSAJ; ISBN: 0-8493-1590-5
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English

AB A review. The pathogenesis of Parkinson's disease remains unknown, even 186 yr after its first description. However, major advances in mol. genetics in the past few decades have revolutionized the classification of Parkinson's disease and Parkinson-

plus syndromes. A strong genetic basis has been established for early-onset Parkinson's disease, and genetic factors have been identified that play a crucial role in a subset of patients with the late-onset, sporadic form. Several genetically, clin., and pathol. distinct forms of these disorders can be caused by mutations in α-synuclein, parkin, UCH-L1, DJ-1, NR4A2, ND4, tau, or as yet unknown causative genes. Mol. characterization has also provided clues regarding their pathogenesis, leading to the categorization of these disorders into polyglutamine disorders, synucleinopathies, and tauopathies. This chapter reviews the current knowledge of the genetic basis of heredofamilial parkinsonism. With growing interest in this field, addnl. genes and susceptibility loci will undoubtedly continue to be discovered.

REFERENCE COUNT: 208 THERE ARE 208 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2005:136592 CAPLUS
DOCUMENT NUMBER: 142:191648
TITLE: Use of a substance that stimulates signaling of human growth hormone receptor in treating Parkinsonism-plus syndrome
INVENTOR(S): Bengtsson, Bengt-Ake
PATENT ASSIGNEE(S): Ares Trading S. A., Switz.
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005014033	A1	20050217	WO 2003-EP50348	20030729
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2532821	A1	20050217	CA 2003-2532821	20030729
AU 2003262552	A1	20050225	AU 2003-262552	20030729
EP 1651250	A1	20060503	EP 2003-817952	20030729
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003018426	A	20060801	BR 2003-18426	20030729
CN 1838966	A	20060927	CN 2003-827096	20030729
JP 2007515375	T	20070614	JP 2005-507528	20030729
ZA 2006000646	A	20070627	ZA 2006-646	20030729
NZ 544695	A	20081128	NZ 2003-544695	20030729
MX 2006000954	A	20060504	MX 2006-954	20060124
KR 2006079183	A	20060705	KR 2006-701726	20060125
NO 2006001004	A	20060426	NO 2006-1004	20060228
US 20070066519	A1	20070322	US 2006-595076	20060907

PRIORITY APPLN. INFO.: WO 2003-EP50348 W 20030729
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to the use of a substance, which binds to and

initiates signaling of the human growth hormone (hGH) receptor or a substance, which stimulates release or potentiates the activity of endogenous hGH, for treatment and/or prevention of Parkinsonism-Plus Syndromes. In particular, the invention relates to the use of hGH for the treatment and/or prevention of Multiple System Atrophy.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2003:708467 CAPLUS
DOCUMENT NUMBER: 140:214854
TITLE: Genetics
AUTHOR(S): Wszolek, Zbigniew K.; Farrer, Matthew
CORPORATE SOURCE: Mayo Clinic, Jacksonville, FL, USA
SOURCE: Neurological Disease and Therapy (2003), 59(Handbook of Parkinson's Disease (3rd Edition)), 325-337
CODEN: NDTHEE; ISSN: 1058-7535
PUBLISHER: Marcel Dekker, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review discusses epidemiol., twin, kindred, and association studies to support the genetic hypothesis of Parkinson's disease (PD) and related parkinsonism plus syndromes. The discovery of mutations in the genes for α-synuclein, ubiquitin C-terminal hydrolase, parkin and tau has created a unique glimpse into the basic mechanisms responsible for neurodegenerative processes.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2001:870863 CAPLUS
DOCUMENT NUMBER: 137:43564
TITLE: Quantitative analysis of striatal dopamine D2 receptors with 123I-iodolisuride SPECT in degenerative extrapyramidal diseases
AUTHOR(S): Prunier, C.; Tranquart, F.; Cottier, J. P.; Giraudeau, B.; Chalon, S.; Guilloteau, D.; De Toffol, B.; Chossat, F.; Autret, A.; Besnard, J. C.; Baulieu, J. L.
CORPORATE SOURCE: Departments of Nuclear Medicine and INSERM U316, University Hospital of Tours, Gif sur Yvette, Fr.
SOURCE: Nuclear Medicine Communications (2001), 22(11), 1207-1214
CODEN: NMCODC; ISSN: 0143-3636
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 123I-Iodolisuride has high specific affinity for binding on dopamine D2 receptors in the striatum and has been used in a few single photon emission computed tomog. (SPECT) studies of extrapyramidal disorders. The diagnosis of Parkinson's disease (PD) is very difficult in the first 5 yr of evolution, with 15-25% false pos. diagnoses. The aim of this study was therefore to determine the value of iodolisuride SPECT in discriminating Parkinson's from the most frequent Parkinson-plus syndromes (PPS). Seventeen patients with an extrapyramidal syndrome had a SPECT examination 1 h after injection of 180-185 MBq of 123I-iodolisuride. They were followed under dopaminergic treatment for at least 2 yr. After 2 yr, they were separated in two groups according to specific clin. criteria and sensitivity to

dopaminergic treatment: nine patients had PD (age = 59.8±8.8 yr; Hoehn and Yahr = 1.8±0.7; evolution = 4.3±3 yr) and eight had PPS (age = 71.6±7.3 yr; Hoehn and Yahr = 2.9±2.0; evolution = 4.1±1.5 yr). The binding potential of iodolisuride in the striatum was assessed by considering the striatum (S)/occipital lobe (O) ratio at the pseudo-equilibrium 1 h after injection. The S/O ratio was statistically different between PD and PPS (1.97±0.3 vs 1.65±0.2 (P<0.02)). Iodolisuride SPECT could differentiate both groups with a sensitivity of 88.8% and a specificity of 75%. Iodolisuride is a good specific D2 receptor ligand for SPECT and complements specific clin. criteria for the diagnosis of Parkinson 's disease and differentiation between different extrapyramidal disorders.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 13 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2001:396192 CAPLUS

DOCUMENT NUMBER: 135:270732

TITLE: Familial Parkinsons' disease and related conditions: Clinical genetics

AUTHOR(S): Wszolek, Zbigniew K.; Uitti, Ryan J.; Markopoulou, Katerina

CORPORATE SOURCE: Department of Neurology, Mayo Clinic Jacksonville, Jacksonville, FL, 32224, USA

SOURCE: Advances in Neurology (2001), 86(Parkinson's Disease), 33-43
CODEN: ADNRA3; ISSN: 0091-3952

PUBLISHER: Lippincott-Raven Publishers

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 55 refs., on the conditions associated with Parkinson 's disease (PD) and the familial forms of parkinsonism, categorized in two groups, i.e., those associated with a PD phenotype and those associated with the parkinsonism-plus syndrome. Topics discussed include: parkinsonian kindreds with known chromosomal loci/mutations; clin., neuropathol. characteristics of the kindreds; and representative kindreds, e.g., family D (Western Nebraska), family H (Greek-American), pallidopontonigral degeneration family, etc.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2001:44415 CAPLUS

DOCUMENT NUMBER: 135:74723

TITLE: Phenotypic correlations in FTDP-17

AUTHOR(S): Reed, L. A.; Wszolek, Z. K.; Hutton, M.

CORPORATE SOURCE: Dept. of Lab. Med. and Pathology, Univ. of Minnesota, Minneapolis, MN, 55455, USA

SOURCE: Neurobiology of Aging (2001), 22(1), 89-107
CODEN: NEAGDO; ISSN: 0197-4580

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 105 refs. Frontotemporal dementias with Parkinsonism linked to chromosome 17 (FTDP-17) are hereditary tauopathies affecting at least 50 known kindred worldwide. Most kindred present with severe behavioral or psychiatric manifestations progressing to dementia, while some kindred first manifest a parkinsonian-

plus syndrome. 9 Missense mutations, 1 deletion mutation, and 2 transition mutations not altering the encoded amino acid, have been described in or near the microtubule-binding domains within exons 9, 10, 12, and 13. In addition, 5 different intronic mutations have been reported in the 5' splice-site of the alternatively spliced exon 10. Missense mutations affecting constitutively expressed exons affect all six major tau isoforms and result in neurofibrillary tangles similar to those present in secondary tauopathies, such as Alzheimer's disease. In contrast, mutations that affect the alternatively spliced exon 10 or its 5' splice regulatory region alter the ratio of the tau isoforms incorporated into the tangles and result in filamentous inclusions resembling those seen in the primary tauopathies, such as progressive supranuclear palsy, corticobasal degeneration, and Pick's disease. The severity and heterogeneity of the clinicomorphol. phenotype may, in part, reflect the diversity in the primary mol. mechanisms of disease in FTDP-17.

OS.CITING REF COUNT: 64 THERE ARE 64 CAPLUS RECORDS THAT CITE THIS RECORD (64 CITINGS)

REFERENCE COUNT: 108 THERE ARE 108 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2000:881185 CAPLUS

DOCUMENT NUMBER: 134:42447

TITLE: Preparation of small cyclic mimics of brain-derived neurotrophic factor (BDNF) and their therapeutic use

INVENTOR(S): Hughes, Richard Anthony; O'Leary, Paul; Zwar, Richard; Hunt-Sturman, Alison

PATENT ASSIGNEE(S): University of Melbourne, Australia

SOURCE: PCT Int. Appl., 104 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000075176	A1	20001214	WO 2000-AU641	20000607
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2376729	A1	20001214	CA 2000-2376729	20000607
EP 1212353	A1	20020612	EP 2000-930886	20000607
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003502295	T	20030121	JP 2001-502457	20000607
AU 780408	B2	20050317	AU 2000-49016	20000607
PRIORITY APPLN. INFO.:			AU 1999-848	A 19990608
			WO 2000-AU641	W 20000607

AB The present invention relates to cyclic compds. comprising one or more cyclic moieties, which have the biol. activity of brain-derived neurotrophic factor (BDNF). Mol. modeling studies led to four peptides cyclo(CEKVPVSKGQLKQC) (L2-12), cyclo(CKVPVSKGQLKC) (L2-10),

cyclo(CVPVSKGQLC) (L2-8), and cyclo(CPVSKGQC) (L-2-6), each constrained by a disulfide bridge between terminal cysteine residues, which were chosen for synthesis and biol. examination The peptides showed a similar pattern of concentration-dependent inhibition of BDNF-mediated survival, causing an increase
in inhibition from 1 x 10-11 to a maximum at approx. 1 x 10-6 M.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 1998:637703 CAPLUS

DOCUMENT NUMBER: 130:33313

TITLE: Early dopaminergic drug-induced hallucinations in parkinsonian patients

AUTHOR(S): Goetz, Christopher G.; Vogel, Caryn; Tanner, Caroline M.; Stebbins, Glenn T.

CORPORATE SOURCE: Department of Neurological Sciences, Rush University/Rush Presbyterian St. Luke's Medical Center, Chicago, IL, 60612, USA

SOURCE: Neurology (1998), 51(3), 811-814
CODEN: NEURAI; ISSN: 0028-3878

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To characterize patients who develop hallucinations early in the course of dopaminergic therapy for Parkinson's disease (PD) and contrast them with patients developing hallucinations after chronic drug treatment. Methods: Parkinsonian patients who met diagnostic criteria for PD, experienced hallucinations, had a detailed hallucination interview at the onset time of their first hallucination, and had a 5-yr clin. follow-up or an autopsy in those 5 yr were identified and divided into two groups for comparison: 12 patients who developed early hallucinations within 3 mo of starting levodopa therapy and 58 PD patients who developed hallucinations after 1 yr of dopaminergic therapy. We contrasted the quality, content, diurnal nature, and emotional elements of the hallucinations, as well as the 5-yr follow-up data on diagnosis, disease course, community home or nursing home outcome, and mortality. Results: Both groups experienced a predominance of visual hallucinations, visions of people and animals, and vivid colors and definition. Features distinctive to the early onset hallucinating patients included visions that persisted in daytime as well as nighttime, frightening content with paranoia, and accompanying nonvisual hallucinations, either auditory, olfactory, tactile, or combinations thereof. At the 5-yr follow-up, none of the early onset hallucinators had PD as their sole disorder. Four of the 12 had an underlying psychiatric illness that included hallucinations or psychosis preceding their parkinsonism by several years. In the other eight patients at the 5-yr follow-up, their parkinsonism evolved to include addnl. signs that were no longer consistent with PD. The primary diagnoses were diffuse Lewy body disease and Alzheimer 's disease (AD) with extrapyramidal signs. Patients with early drug-induced hallucinations had significantly greater placement to nursing homes and greater mortality. Conclusions: Early onset drug-related hallucinations are not typical of PD. Their presence should signal an investigation of two alternative diagnoses, either a comorbid psychotic illness (often unrevealed by the patient initially) or an evolving parkinsonism-plus syndrome.

OS.CITING REF COUNT: 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 17 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 1998:409143 CAPLUS
DOCUMENT NUMBER: 129:174113
ORIGINAL REFERENCE NO.: 129:35364h,35365a
TITLE: Loss of dopamine-D2 receptor binding sites in parkinsonian plus syndromes
AUTHOR(S): Hierholzer, Johannes; Cordes, Michael; Venz, Stephan; Schelosky, Ludwig; Harisch, Cordula; Richter, Wolf; Keske, Uwe; Hosten, Norbert; Maurer, Jurgen; Poewe, Werner; Felix, Roland
CORPORATE SOURCE: Strahlenklinik und Nuklearmedizinische Klinik der Charite der, Humboldt-Universitat zu Berlin, Berlin, 13353, Germany
SOURCE: Journal of Nuclear Medicine (1998), 39(6), 954-960
CODEN: JNMEAQ; ISSN: 0161-5505
PUBLISHER: Society of Nuclear Medicine
DOCUMENT TYPE: Journal
LANGUAGE: English
AB This study analyzed temporal changes of striatal dopamine-D2 receptor binding during the course of different extrapyramidal movement disorders using 123I-iodobenzamide (IBZM) SPECT. Eighteen patients (9 with Parkinson's disease, 9 with parkinsonian plus syndrome) were followed for 11-53 mo. Dopamine-D2 receptor binding was assessed using 123I-IBZM SPECT at the beginning and at the end of the follow-up period. SPECT data were acquired 120 min postinjection of 3-5 mCi 123I-IBZM. A semiautomated algorithm was applied to the raw data for semiquant. evaluation of regional cerebral receptor binding. Intraobserver (r = 0.992) and interobserver (r = 0.930) variance was low for the semiautomated interpretation of the SPECT examination of the dopaminergic D2 receptor binding, reflecting a highly reproducible SPECT algorithm. Mean specific dopamine-D2 receptor binding was lower in patients with parkinsonian plus syndrome compared to patients with Parkinson's disease on the initial ($p < 0.001$) as well as the follow-up study ($p < 0.001$). In patients with Parkinson's disease, we observed an unaffected receptor binding compared to a reduced binding of radiotracer in patients with parkinsonian plus syndrome during the course of the disease ($p < 0.001$). During the follow-up, patients with Parkinson's disease showed a constant dopamine-D2 receptor binding. In contrast, patients with parkinsonian plus syndrome revealed a decline of the binding of dopamine-D2 receptor. These findings are in agreement with histopathol. data that demonstrated a preserved dopamine-D2 receptor status in patients with Parkinson's disease and a decline of the dopamine-D2 receptors in patients with parkinsonian plus syndrome. SPECT examns. using 123I-IBZM are useful for assessing dynamic changes of dopamine-D2 receptors in extrapyramidal movement disorders. Semiquant. SPECT evaluations may provide valuable information for clin. management and prognosis of the patient with extrapyramidal movement disorders.
OS.CITING REF COUNT: 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 1997:258880 CAPLUS
DOCUMENT NUMBER: 126:274240
ORIGINAL REFERENCE NO.: 126:53077a,53080a
TITLE: IBZM- and CIT-SPECT of the dopaminergic system in parkinsonism
AUTHOR(S): Tissingh, G.; Booij, J.; Winogrodzka, A.; Van Royen,

E. A.; Wolters, E. Ch.
CORPORATE SOURCE: Department of Neurology, Graduate School Neurosciences Amsterdam, Academisch Ziekenhuis VU Amsterdam, Amsterdam, Neth.
SOURCE: Advances in Research on Neurodegeneration (1997), 5, 31-37
CODEN: ARNEFX; ISSN: 1068-719X
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Parkinsonism is most of the time caused by idiopathic Parkinson's disease (IPD). Considering the differences in therapeutic response and prognosis, in vivo discrimination between IPD and "parkinsonism-plus" syndromes is important. Recently, ligands have become available for imaging the pre- and postsynaptic dopaminergic system by Single Photon Emission Computed Tomog. (SPECT). Visualization of postsynaptic D2 dopamine receptors using 123I-iodobenzamide (123I-IBZM) may contribute to the differential diagnosis between IPD and "parkinsonism-plus" syndromes as IPD is a pure presynaptic disease. Imaging of the presynaptic dopamine transporters using [123I]β-CIT (2β-carbomethoxy-3β-(4-iodophenyl)tropane) may be used as a diagnostic technique. Early disease detection in subjects suspected to be at risk for developing IPD has become possible using [123I]β-CIT or other ligands for the dopamine transporter. Furthermore, with SPECT one is probably able to monitor in an objective way the efficacy of new pharmacol. therapies.